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Research Paper

Current status of *Escherichia coli* strain infections and their diagnosis

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ABSTRACT

Escherichia coli infection is contagious and can be spread from one individual to another through fecal contagion. Contaminations that produce later normal symptoms may require a group of experts to give care and diagnosis of the long-suffering person is absolutely made when *E. coli* 0157: H7 is isolated usually from the sick person or animal stool and recognized with immunologic examinations. Most *E. coli* 0157: H7 contagions resolute instinctively and need no cure. However, helpful cure is rapidly required if the person or animal becomes dehydrated and anemic. Contamination with the bacteria is typically derived from consumption of unclean food. Inhibition of contamination lies in the consumption healthy cooked foods, particularly hamburger, and drinking preserved or pasteurized fluids. Escaping moving or eating any food that may be contaminated with any animal or human waste will help stop the infection. This review discusses the current status of the pathogenesis of diarrheagenic *E. coli* strains. The clinical manifestations, diagnosis and epidemiologic investigation of these strains are described. Additionally, people are advised not to consume contaminated foods. Farmers of gazelles and other susceptible animals are advised to maintain a sufficient distance from or isolate these animals to avoid fecal contamination.

Key words: Cattle, *Escherichia coli*, Gazelle, human.

INTRODUCTION

Enterotoxigenic *Escherichia coli* (ETEC) and other pathogens

Humans working in the field of food processing and milk products may contaminate the food with *Streptococcus pneumoniae*. Its common host is the human body, in which it often does not cause disease but at other times it can cause diseases specifically, pneumonia, otitis media, bacteremia, meningitis, peritonitis and sinusitis. The emergence of infection caused by invasive penicillin non-susceptible (PNS) and multidrug-resistant strains of *S. pneumonia* has become a worldwide concern, necessitating the epidemiologic surveillance of such strains and other strains of *E. coli* (Al-Swailem et al., 2004).

Arcanobacterium (*Actinomyces)* pyogenes are animal pathogens; they produce hemolytic exotoxin, pyolysin (PLO) and are opportunistic pathogens of economically important livestock such as dairy, beef cattle and sheep (Omar, 2012). The studies of Enterotoxigenic *E. coli* (ETEC) have demonstrated that Enterotoxigenic *E. coli* cause diarrhea in infants younger than 2 years of age. Cases in Egypt account for approximately 70% of the first episodes of ETEC; the incidence was found to be higher in males than in females.

In different countries where ETEC is endemic, characteristics such as toxin type and colonization factors of ETEC differ. A potential contributor to the lack of attention to the epidemiology of EIEC is that it is often observed to be an infrequent cause of diarrhea relative to other diarrhea-causing *E. coli* (Nadia et al., 2007). Most

travelers, children and adults living in developing countries with proper sanitation and clean water are less affected by ETEC and are thus protected from diarrhea. Heat stabile and heat labile enterotoxins of ETEC were previously identified and the characterization, sequencing, cloning and genetic control in transmissible plasmids previously evaluated (Firdausi et al., 2005).

PREVALENCE AND PATHOGENICITY OF *ENTEROPATHOGENIC E. COLI* (EPEC)

Myron (1987) reported that EPEC have various mechanisms of pathogenesis depending on the presence of specific genes. The eae, bfpA and bundle-forming pilus genes were used for the identification of EPEC and subdivision of this group of bacteria into typical and atypical strains.

Atypical-EPEC (aEPEC) is more prevalent than typical-EPEC (tEPEC) in both developed and developing countries and is important in both pediatric endemic diarrhea and diarrhea outbreaks. The virulence mechanisms and pathogenicity of the attaching and effacing lesion (A/E) and type-three-secretion-system (T3SS) are important for these strains. Attaching and effacing lesions of the pool locus genes are used for attachment, while enterocyte effacement (LEE)-encoded and non- LEE-encoded effector proteins are used to subvert and modulate cellular and barrier properties of the host (John et al., 2011).

Shiga toxin-producing *E. coli* (STEC)

These strains have emerged as significant causes of human disease. Five years before their identification as pathogens, STEC strains belonging to the serotype O26: H11 and STEC O26 were recognized for their production of Stx1 and Stx2 (Zhang et al., 2000; Dean et al., 2008). Verocytoxin strains produce Shiga toxin and is associated with food-borne zoonotic outbreaks worldwide (Nguyen et al., 2012; García et al., 2007; Dunn et al., 2004). Myron et al. (1987) classified a variety of Stx producing *E. coli* O26: H11 strains as enterohemorrhagic *E. coli* (EHEC). Verocytotoxin producing *E. coli* (VTEC) emerged as foodborne pathogens that can cause severe and potentially fatal illnesses, such as hemorrhagic colitis and hemolytic uremic syndrome (Bonardi et al., 2007). An agricultural fair-associated

Shiga-toxigenic *E. coli* (STEC) outbreak was unusual in that it affected both livestock exhibitors and visitors. Livestock exhibitors and fair visitors should follow guidelines to reduce the risk of transmission of STEC at agricultural fairs (Durso et al., 2005). The presence of *E. coli* at county fairs suggests the potential for transmission to the public, who may have contact with cattle or the livestock environment (Cho, 2006). Unlike many gastro-intestinal pathogens, EPEC can modulate host cellular and immune responses from the exterior of the infected cell, chiefly through the secreted and translocated components of a type III secretion system. Close inspection of these EPEC proteins and the interactions they mediate provided an increasingly coherent picture of the pathogenic mechanisms by which EPEC exploit their hosts (Delahay et al., 2001). Bacterial pathogenicity islands (PAI) often encode both effector molecules responsible for the disease and secretion systems that deliver these effectors to host cells (Deng et al., 2004).

Enter invasive *E. coli* (EIEC) and enter aggregative *E. co*li (EAEC)

EIEC were first discovered in 1971 and found to cause diarrheal disease. These bacteria cause shigellosis-like symptoms in adults and children. Very few studies have attempted to identify the individual risk factors for infection, possible reservoirs, and infection rates by these strains. Specific endospore formers have become important contaminants in industrial food processing (Oamr, 2014). Air-conditions (ACS) contamination is a major complication following living in hot parts of the world. Microbial contamination of unclean ACS is a precursor for infection (Omar, 2013).

Contaminated groundwater supplies used for public or private water supplies can result in outbreaks of disease that are more prevalent in under developed countries (Ingrid, 2014). The use of waste water in agriculture and aquaculture is a sensitive subject. Microbial contaminants in cucumber from the use of sewage water, the air during harvest and transportation stayed in cucumber (Omar et al., 2014). EAEC form a subgroup of diarrheagenic *E. coli* with the ability to adhere to epithelial cells such as HEp-2 in a very characteristic stacked-brick pattern. Numerous studies to identify specific virulence factor(s) unique for this category have been published, but it remains unknown how EAEC cause persistent diarrhea.

Additionally, the aggregative property of EAEC makes serotyping difficult because the cells auto-agglutinate (Weintraub, 2007). At least five categories of diarrheagenic *E. coli* strains were recognized based on distinct epidemiological and clinical features, specific virulence determinants and associations with certain serotypes (Isabel et al., 2002). Virulence factors phenotypic and genotypic characteristics and relatedness of *E. coli* O157: H7 strains have very strong virulence factors contributing to their low infectious dose and these bacteria survive well under different conditions, such as low pH. Among the genes on the LEE locus are eae (originally known as eaeA), which encodes intimin, an outer membrane protein required for these strains to adhere to mammalian cells (Gansheroff et al., 2000).

The immunomagnetic separation procedure for sorbitol-fermenting (SF) STEC strains isolated from two patients

were compared; one patient had hemolytic-uremic syndrome and the other had diarrhea. The phenotypic and genotypic characteristics of all isolates were identical or closely related (Bielaszewsk et al., 2000). Attaching and effacing strains have a similar mode of interaction with epithelial cells: following initial adherence to epithelial cell surfaces, they exert their pathological effects by causing histopathological alterations termed A/E lesions (Zhang et al., 2002). STEC and VTEC strains are the most important recently emergent group of food-borne pathogens. These bacteria can cause severe disease in humans, such as hemorrhagic colitis and hemolytic uremic syndrome (Blanco et al., 2004).

IDENTIFICATION OF HEMORRHAGIC AND DIARRHEA-CAUSING STRAINS

EHEC strains cause hemorrhagic gastro-intestinal disease and hemolytic uremic syndrome (Caprioli et al., 2005; Isabel et al., 2002). Using sorbitol-MacConkey (SMAC) agar, only O157: H7 was detected (Novicki et al., 2000). Strains TT12A and TT12B have the same biochemical profiles in AP120E analysis and both showed characteristic traits of O157: H7 strains in that they grew on CT-SMAC medium and did not ferment sorbitol or exhibit GUD activity. Therefore, these two strains were indistinguishable by standard biochemical assays used to identify O157: H7 strains (Feng et al., 2001). Microbiological diagnostic procedures based on the characteristic phenotypic feature of this pathogen, particularly its inability to ferment sorbitol after overnight incubation revealed that new strains of the serotype were non-motile, ferment sorbitol rapidly and cause human diseases (Karch et al., 2001).

NEW FIELD OF DNA SEQUENCE ANALYSIS

Horizontal genomics is a new field in prokaryotic biology focused on the analysis of DNA sequences in prokaryotic chromosomes that appear to have originated from other prokaryotes or eukaryotes (Frost et al., 2005). Polymerase chain reaction (PCR) is a very rapid and reliable tool for molecular biology-based diagnosis of a variety of infectious diseases. Fecal samples are the most difficult specimens to test by direct PCR due to the presence of inherent PCR inhibitors that are often extracted along with bacterial DNA (Holland et al., 2000). Several commercial methods and within-laboratory methods were evaluated, including the ISOQuick kit and the QIAamp kit. The in-house enrichment method includes a 4-h tryptic soy broth enrichment of stool, followed by heat lysis with Triton X-100 lysis buffer. The non-uniformity of stool samples in parameters such as physical matter, target organisms and associated background fecal flora makes extraction of DNA from fecal specimen highly variable between specimens in terms both the yield and purity. False-negative PCR results may occur as a result of the presence of a small number of target organisms in the volume of stool sampled and decrease in stability of cells during storage (Kalita et al., 2014).

MAJOR RESERVOIR AND SOURCE OF INFECTION

Meyer et al. (2001) reported that cattles were major reservoirs and sources of infection. Consumption of undercooked meat products from cattle has been associated with many human infections and the O157: H7 strains have been detected in bovine feces throughout the United States, Canada and Europe. The possible role of wild deer in the epidemiology of these strains received attention as early as 1988 when the strains were isolated from undercook deer meat (Fisher et al., 2001; Naylor et al., 2003). Healthy sheep are a major reservoir of STEC pathogens for humans (Blanco, 2003). These Shiga-toxigenic strains were isolated from tissues in the cattle gastro-intestinal tract, including the tonsils, reticulum, rumen, omasum, abomasum, duodenum, jejunum, cecum, spiral colon, rectum and even the liver, suggesting that Shiga-toxigenic strains were largely adapted to several cattle gastro-intestinal microhabitats.

Recent studies have shown conflicting results regarding the bovine gallbladder as a focus of colonization in cattle (Keen et al., 2002). Many Shiga-toxigenic strain outbreaks related to food were of bovine origin, including infections owing to the consumption of ground beef, roast beef and raw milk and foods likely contaminated by bovine feces, such as lettuce and alfalfa. Nonfood- borne outbreaks have been associated with attending child day care, drinking contaminated water and swimming in unchlorinated water (Kassenborg et al., 2004; Kelly et al., 2009).

Six pathogenic groups and their clinical signs

There are six pathogenic groups that may cause diarrhea: ETEC, EHEC, EIEC, EPEC, EAEC and diffusely adherent groups (Rodríguez et al., 2002) (Table 1). These strains are a threat to public health and have been implicated in many outbreaks of hemorrhagic colitis, some of which include fatalities caused by hemolytic uremic syndrome (Perna et al., 2001). The human pathogenic strains cause hemorrhagic colitis and transiently colonize healthy cattle at the terminal rectal mucosa (Sheng et al., 2006).

EHEC infections in humans are an important public health issue and are commonly acquired through contact with ruminant feces (Van et al., 2005). Infection with *E. coli* strains cause an estimated 70,000 instances of diarrheal illness per year in the United States and can result in hemolytic-uremic syndrome and death. Environmental

Table 1: Pathogenicity and clinical manifestations of six *E. coli* strains.

Strains	Pathogenicity	Clinical manifestation
ETEC	Produces enterotoxin and remains active at higher temperature.	Acute watery diarrhea and Afebrile.
EAEC	Small and large bowel adherence and forms pores.	Watery and bloody diarrhea.
EPEC	Adheres to Small bowel and epithelial cell mediated by virulence factor adhesion of EPEC	Severe acute watery diarrhea and bloody diarrhea.
EIEC	Adheres to mucosa of gastro-intestinal and causes inflammation of large bowel.	Watery diarrhea, dysentery like diarrhea and fever.
DAEC	Diffuses and adheres to epithelial cells.	Watery diarrhea.
STEC	Adheres to Large bowel by virulence factor adhesion of EPEC; Shiga toxin 1, Shiga toxin 2 production.	Watery diarrhea that often progresses to bloody diarrhea.

contamination with *E. coli* strains may be a public health problem (Varma et al., 2003). The reuse of waste water in agriculture and aquiculture is a sensitive subject. Great care must be taken when introducing these techniques. Potential health risk with use of sewage as fertilizer for irrigation and its performance were highlighted (Omar, 2010). These bacteria are recognized worldwide, particularly in developed countries, as emerging food-borne bacterial pathogens that cause disease in humans and in some animals (Yoon and Hovde, 2008). *E. coli* strains are versatile bacterial species that include harmless commensal and different pathogenic variants with the ability to both cause intestinal or extra intestinal diseases in humans and many animal hosts (Leimbach et al., 2013). The prevalence of EHEC in cattle peaks in the summer and is higher in post-weaned calves and heifers than in younger and older animals (Farfan et al., 2012; Dziva et al., 2007). Virulent strains of EHEC are rarely harbored by pigs or chickens, but are found in turkeys (Zumbrun et al., 2013).

GASTRO-INTESTINAL TRACT COLONIZATION

The mammalian gastro-intestinal tract is colonized by a group of bacterial species. Bacteria engage in chemical signaling to coordinate population-wide behavior. However, it is unclear whether chemical sensing plays a role in establishing mammalian host bacterial commensal relationships (Hughes et al., 2010). The Toll-like receptor interleukin 1 receptor signaling receptor superfamily is important in differentially recognizing pathogen products and in eliciting appropriate immune responses. These receptors alter gene expression mainly by activating nuclear factor-kappaB and activating protein 1. Single immunoglobulin interleukin-1R-related molecule, a member of this family that does not activate these factors negatively modulates immune responses (Wald et al., 2003; Sham et al., 2013; Ochoa et al., 2008).

INTIMIN AND INTESTINAL COLONIZATION

Some strains of *E. coli* carry a large virulence plasmid that encodes the etp type II secretion system, which secretes the genetically linked zinc metalloprotease StcE. The Ler regulator controls the expression of many genes involved in A/E lesion formation, including StcE, indicating that StcE is important at a similar time during colonization and that exogenous recombinant StcE increases close adherence. Thus, StcE may help block host clearance of the pathogenic organism through destruction of some classes of glycoproteins and may contribute to the close adherence of agents on the HEp-2 cell surface (Grys et al., 2005; Reinstein et al., 2007).

Intimin facilitates intestinal colonization through enterohemorrhagic agents; however, the importance of intimin binding to its translocated receptor (Tir) as opposed to cellular coreceptors is unknown. The intimin-Tir interaction is required for optimal actin assembly under adherent bacteria *in vitro*, a process which requires the Tir-cytoskeleton coupling protein (TccP/EspF(U)) (Vlisidou et al., 2006). Most loci of enterocyte effacement (LEE) genes were upregulated in TW14359, whereas flagellar and chemotaxis genes were primarily upregulated in Sakai, indicating discordant expression of these genes between the two strains. Shiga toxin 2 genes were also upregulated in the TW14359 strain, as several strains encoding genes that promote adherence including type II secretion genes and their effectors (Abu-Ali et al., 2010).

EFFECT OF BACTERIOPHAGES AGAINST *E. COLI*

Some studies investigated the phages e11/2 and e4/1c against *E. coli* in an *ex vivo* rumen model and in cattle *in vivo*. In the *ex vivo* rumen model, samples were inoculated with either 10^3 or 10^6 CFU/ml inoculum of *E. coli* and challenged separately with each bacteriophage. In the presence of phage e11/2, the numbers of *E. coli* O157: H7

bacteria were significantly reduced to below the limit of detection within 1 h (P= 0.05). Phage e4/1c significantly reduced *E. coli* numbers within 2 h of incubation (P= 0.05), but the number of surviving *E. coli* bacteria remained unchanged over a further 22 h incubation period (Rivas et al., 2010). A guanine-to-thymine transversion in the csgD promoter of *E. coli* strain ATCC 43895 led to the creation of strain 43895OR, which produces abundant extracellular matrix rich in curly fibers, forms biofilms on solid surfaces, invades cultured epithelial cells and is more virulent in mice compared to strain 43895 (Uhlich et al., 2009).

BACTERIAL GENOME EVOLUTION

E. coli are considered model organisms for analyzing the processes involved in bacterial genome evolution, as the species is comprised of numerous pathogenic and commensal variants. Pathogenic and non-pathogenic strains differ in the *presence E. coli* and absence of additional DNA elements contributing to specific virulence traits and in the presence and absence of additional genetic information (Dobrindt et al., 2003). Experimentally inoculated sheeps and cattles were used as models of natural ruminant infection to investigate the pattern of *E. coli* shedding and gastro-intestinal tract location. The predominant location of *E. coli* persistence was the lower gastro-intestinal tract. *E. coli* was rarely cultured from the rumen or duodenum after the first week post-inoculation, but this did not predict whether animals shed the bacteria at the end of the one week or one month. These findings indicate that the colon is the site of *E. coli* persistence and proliferation in matured ruminant animals (Grauke et al., 2002).

To determine whether *E. coli* resides in the gall bladder of cattle, inoculation studies were conducted with strain 86 to 24 in weaned Holstein calves. Strain 86 to 24 was isolated from the gall bladders of five calves at 36 days after inoculation. Two other calves contained the inoculation strain in the distal colon, but the organism was absent in their gall bladders. These studies showed that *E. coli* can reside transiently or permanently at a low level in the gall bladder of cattles (Jeong et al., 2007). Cattles consistently positive for *E. coli* cultures for a long duration were euthanized and necropsied. Tissue from along the gastro-intestinal tract were cultured for the bacteria and examined histologically for a lymphoid character. *E. coli* was detected only at the recto anal junction mucosa and not at any other gastro-intestinal tract location (Lim et al., 2007).

IMPORTANT GENES FOR ADHERENCE

Expression of genes of the LEE is essential for adherence of EHEC to intestinal epithelial cells. Gut f actors that modulate LEE gene expression may influence the outcome of the infection (Branchu et al., 2014). EHEC O157: H7, in the context of virulence factor serotypes are global zoonotic pathogens responsible for most severe cases of human EHEC disease. EHEC strains are carried primarily by healthy cattles and other ruminants, but most bovine strains are not transmitted to people and do not exhibit virulence factors associated with human disease. The prevalence of EHEC is likely underestimated. Carriage of EHEC by individual animals is typically short-lived, but pen and farm prevalence of specific isolates may extend for months or years and some carriers designated as super shedders may harbor high intestinal numbers of the pathogen for extended periods (Ferens and Hovde, 2011).

The definition of EAEC is the ability of the micro-organism to adhere to epithelial cells such as HEp-2 cells in a very characteristic 'stacked-brick' pattern. Although many studies searching for specific virulence factor(s) unique for this category of DEAC have been published, it remains unknown why EAEC cause persistent diarrhea. In addition, the aggregative property of EAEC causes numerous issues in serotyping due to auto-agglutination of the cells (Weintraub, 2007).

MORBIDITY AND MORTALITY ASSOCIATED WITH EHEC

The EHEC serotype is the cause of bloody diarrhea and hemolytic uremic syndrome worldwide. Antimicrobials trigger the SOS response in EHEC to promote the release of the potent Shiga toxin that is responsible for much of the morbidity and mortality associated with EHEC infection (Nguyen and Sperandio, 2012). Fifty-seven *E. coli* strains isolated from patients in six countries were investigated by PCR restriction fragment length polymorphism (RFLP) analysis of the flagellin-encoding (fliC) gene (fliC RFLP analysis). The strains were determined by serotyping and found to belong to five different types or were non-motile. The fliC RFLP analysis revealed only two different patterns among the 57 strains. One fliC RFLP pattern was displayed by 54 strains and was identical to that of the *E. coli* H11 reference strain Su4321-41. The other fliC RFLP pattern was observed for three strains and found identical to that of the *E. coli* H32 reference strain K10 (Zhang et al., 2000).

Enrichment and direct non-enrichment recto anal mucosal swab (RAMS) culture techniques were developed and compared to traditional fecal culture for the detection of *E. coli* in experimentally infected and naturally infected cattles. RAMS culture predicted the duration of the infection. Cattle transiently shedding *E. coli* for one week were positive by fecal culture only and not by RAMS culture, whereas colonized animals (which were culture-positive for an average of 26 days) were positive early by RAMS culture. RAMS culture more directly measured the relationship between cattles and *E. coli* infection compared

to fecal culture (Rice et al., 2003). Fifteen weaned calves (age 89 to 141 days) were treated with dexamethasone (0.25 mg/kg, IV) for 3 days before, on the day of inoculation and after inoculation with 10 colony-forming units of either *E. coli* strain 86-24, which produces Shiga toxin 2 and intimin (n = 13) or non-pathogenic *E. coli* (strain 123, which does not produce Shiga toxin or intimin (n = 2). All calves were necropsied 4 days after inoculation and histologic lesions of attaching and effacing bacteria observed in the large intestine (12/13) and gallbladder mucosa (5/13) of calves inoculated with *E. coli* 86- 24. Cholecystitis was present in 12 of 13 calves that received *E. coli* 86-24. Inoculum bacteria were recovered from the distal colons or feces (13/13) and gallbladders (3/4) of calves inoculated with 86-24 (Stoffregen et al., 2004).

The frequency of STEC serotypes associated with post-diarrheal hemolytic uremic syndrome cases among children and adults in the United States and proportion with IgM or IgG lipopolysaccharide antibodies to *E. coli* was determined using a nationwide sample from January, 1987 through December, 1991. Among the 83 patients, STEC was isolated from 30 (43%) of 70 patients whose stool cultures yielded bacterial growth (25 *E. coli* isolates and 5 non-STEC isolates). 53 (80%) of 66 patients with serum samples showed positive lipopolysaccharide antibody titers. Of the 83 patients, 60 (72%) had evidence of STEC infection, including 6 of 8 adults whose illnesses also met the criteria for thrombotic thrombocytopenic purpura (Banatvala et al., 2001). Different controlling attitudes between livestock owners, animal health authorities and wildlife conservationists are largely associated with wildlife (Bengis et al., 2002). White-tailed deer (*Odocoileus virginianus*) have been implicated in the transmission of this bacterium to humans and were suggested as reservoirs affecting carriage in cattle populations (John et al., 2004). Gastroenteritis morbidity is high among children under the age of 4 years, particularly, those who attend day care (Enserink et al., 2014). Fecal shedding of *E. coli*, the prevalence of *E. coli* in pens and on carcasses and hides and cattle performance following daily dietary supplementation with lactobacillus-based direct-fed microbials were evaluated in a feeding trial involving 180 beef steers. Steers were evaluated for shedding of *E. coli* by an immunomagnetic separation technique upon arrival at the feedlot, just before treatment with the direct-fed microbials and every 14 days thereafter until slaughter (Brashears et al., 2003).

EHEC constitutes a subset of serotypes (*E. coli* O157 and some other serogroups) of STEC firmly associated with severe human illnesses such as bloody diarrhea and hemolytic uremic syndrome. Stx production is essential but not sufficient for EHEC virulence. Most strains are capable of colonizing the intestinal mucosa of the host through an A/E mechanism, genetically governed by a large PAI defined as the LEE. Other virulence factors carried by mobile genetic elements such as PAI and plasmids were recently described and their role in the pathogenic process is not fully understood.

EHEC are zoonotic pathogens that rarely cause disease in animals and ruminants are recognized as their main natural reservoir (Putonti et al., 2006). Foodborne outbreaks are defined as the occurrence of ≥2 cases of *E. coli* infection resulting from ingestion of a common food, or if food vehicle was undetermined, sharing a common meal or food facility. Food vehicles were grouped into the following categories: ground beef, other beef products and dairy. Food vehicles were found to be a significant vehicle in case-control studies ($p \leq 0.05$) by isolation of *E. coli* from a suspected item or by being the only common food item consumed. A multistate outbreak is defined as exposure to a common vehicle occurring in one state. Hemolytic uremic syndrome cases were classified by individual investigators and included cases diagnosed as thrombotic thrombocytopenic purpura following *E. coli* infection (Rangel et al., 2005).

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